

U.S. Patent Appl. No. 09/019,441  
Attorney Docket No. 037003-0275470

Amendments To The Specification

Please amend the paragraph beginning at page 9, line 27, as follows:

Figure 9 compares the *in vivo* inhibitory activity of primate anti-human 6G5 and a PRIMATIZED® version thereof p6GSG4P p6GSG4P.

Please amend the paragraph beginning at page 18, line 4, as follows:

The PRIMATIZED® gamma 1 version of primate 6G5 was found to inhibit induced IgE expression in SCID mice while the same concentration of either the primate 6G5 or the PRIMATIZED® p6G5F4p p6G5G4p did not inhibit induced IgE expression. Therefore, an antibody containing human gamma-1 constant domains was found to be even more effective in an *in vivo* animal model than than the primate monoclonal antibody. Furthermore, the inventors anticipate that anti-CD23 antibodies containing human gamma-3 constant domains will be just as effective as those having gamma-1 constant domains, because gamma-1 and gamma-3 constant domains have affinity for the same classes of Fc receptors.

Please amend the paragraph beginning at page 34, line 13, as follows:

The radio-activity radioactivity counts in each well are then determined by running the wells on a gamma counter.

Please amend the paragraph beginning at page 35, line 20, as follows:

The radio-activity radioactivity counts in each well are then determined by running the wells on a gamma counter.

Please amend the paragraph beginning at page 55, line 20, as follows:

Based upon the sequence of 5E8 heavy variable domain, there is a potential glycosylation site of the immunoglobulin at asparagine codon 75. This potential glycosylation site corresponds to a conserved asparagine-linked glycosylation motif having the following tripeptide sequence: (Asp) (Asn) - (Any amino acid except proline) - (Serine or threonine). Therefore, a glycosylation mutant of 5E8, which would be unable to be glycosylated at this position because of modification of this glycosylation motif, was generated by replacing the asparagine codon 75 with a lysine (which is found in many human immunoglobulins at this position). Site specific mutagenesis was effected by the following methods.

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Please amend the paragraph beginning at page 62, line 2, as follows:

For example, the regions of the IgG constant region involved in FcR binding and interaction with the C1q complement component have been characterized, and mutations have been identified which either increase or decrease binding affinity. As disclosed in U.S. Patent 5,648,260, herein incorporated by reference, changing Leu 235 to Glu in the human IgG3 constant region destroys the interaction of the mutant for the human Fc gamma RI receptor. Furthermore, mutations on adjacent or close sites in the hinge link region (i.e. replacing residues 234, 235, 236 or 237 by Ala) indicate that alterations in residues 234, 235, 236 and 237 at least affect affinity for the Fc gamma RI receptor.

Please amend the sequence at page 69, line 5, as follows:

The three primate antibodies (p5E8G4P, p5E8G4PN-, and pGG5G4P p6G5G4P) were then expressed as human gamma-1 versions using substantially the same methodology. All three human gamma-1 anti-human CD23 antibodies, respectively designated p5E8G1, p5E8G1N- and pGG5G1 p6G5G1, were found to be active in the *in vitro* IL-4/IgE assay (Figures 3 and 5).